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FILE 'HOME' ENTERED AT 13:49:27 ON 10 JAN 2006

=> file medline  
COST IN U.S. DOLLARS                      SINCE FILE              TOTAL  
   ENTRY              SESSION  
FULL ESTIMATED COST                      0.21              0.21

FILE 'MEDLINE' ENTERED AT 13:49:44 ON 10 JAN 2006

FILE LAST UPDATED: 7 JAN 2006 (20060107/UP). FILE COVERS 1950 TO DATE.

=> s uncoupl?  
L1        15992 UNCOUPL?

=> s l1(p) receptor#  
          725556 RECEPTOR#  
L2        2638 L1(P) RECEPTOR#

=> s l2(p)G  
          556021 G  
L3        780 L2(P)G

=> s l3(p) ident?  
          1076690 IDENT?  
L4        100 L3(P) IDENT?

=> d 1-100 ti so

L4    ANSWER 1 OF 100        MEDLINE on STN  
TI    Hydrogen peroxide causes uncoupling of dopamine D1-like receptors from G  
      proteins via a mechanism involving protein kinase C and G-protein-coupled  
      receptor kinase 2.  
SO    Free radical biology & medicine, (2006 Jan 1) 40 (1) 13-20. Electronic  
      Publication: 2005-09-08.  
      Journal code: 8709159. ISSN: 0891-5849.

L4    ANSWER 2 OF 100        MEDLINE on STN  
TI    Human adenosine A(3) receptor leads to intracellular Ca(2+) mobilization  
      but is insufficient to activate the signaling pathway via phosphoinositide  
      3-kinase gamma in mice.  
SO    Biochemical pharmacology, (2005 Nov 15) 70 (10) 1487-96. Electronic  
      Publication: 2005-09-12.  
      Journal code: 0101032. ISSN: 0006-2952.

L4    ANSWER 3 OF 100        MEDLINE on STN  
TI    Osteoblast calcium-sensing receptor has characteristics of ANF/7TM  
      receptors.  
SO    Journal of cellular biochemistry, (2005 Aug 15) 95 (6) 1081-92.  
      Journal code: 8205768. ISSN: 0730-2312.

L4    ANSWER 4 OF 100        MEDLINE on STN  
TI    Self-limitation of intravenous tocolysis with beta2-adrenergic agonists is  
      mediated through receptor G protein uncoupling.  
SO    Journal of clinical endocrinology and metabolism, (2005 May) 90 (5)  
      2882-7. Electronic Publication: 2005-02-22.  
      Journal code: 0375362. ISSN: 0021-972X.

L4    ANSWER 5 OF 100        MEDLINE on STN  
TI    Estrogen modulation of hypothalamic neurons: activation of multiple  
      signaling pathways and gene expression changes.  
SO    Steroids, (2005 May-Jun) 70 (5-7) 397-406. Electronic Publication:  
      2005-04-21. Ref: 127  
      Journal code: 0404536. ISSN: 0039-128X.

L4    ANSWER 6 OF 100        MEDLINE on STN  
TI    The natural mutation encoding a C terminus-truncated 5-hydroxytryptamine-  
      2B receptor is a gain of proliferative functions.  
SO    Molecular pharmacology, (2005 Apr) 67 (4) 983-91. Electronic Publication:  
      2004-12-29.  
      Journal code: 0035623. ISSN: 0026-895X.

L4    ANSWER 7 OF 100        MEDLINE on STN  
TI    Antiepileptic popular ketogenic diet: emerging twists in an ancient story.

SO Progress in neurobiology, (2005 Jan) 75 (1) 1-28. Electronic Publication:  
2005-01-27. Ref: 220  
Journal code: 0370121. ISSN: 0301-0082.

L4 ANSWER 8 OF 100 MEDLINE on STN  
TI Interaction between mGluR8 and calcium channels in photoreceptors is  
sensitive to pertussis toxin and occurs via G protein betagamma subunit  
signaling.  
SO Investigative ophthalmology & visual science, (2005 Jan) 46 (1) 287-91.  
Journal code: 7703701. ISSN: 0146-0404.

L4 ANSWER 9 OF 100 MEDLINE on STN  
TI Regulation of corticotropin-releasing hormone receptor type 1alpha  
signaling: structural determinants for G protein-coupled receptor  
kinase-mediated phosphorylation and agonist-mediated desensitization.  
SO Molecular endocrinology (Baltimore, Md.), (2005 Feb) 19 (2) 474-90.  
Electronic Publication: 2004-10-21.  
Journal code: 8801431. ISSN: 0888-8809.

L4 ANSWER 10 OF 100 MEDLINE on STN  
TI Peptide ligand binding properties of the corticotropin-releasing factor  
(CRF) type 2 receptor: pharmacology of endogenously expressed receptors,  
G-protein-coupling sensitivity and determinants of CRF2 receptor  
selectivity.  
SO Peptides, (2005 Mar) 26 (3) 457-70.  
Journal code: 8008690. ISSN: 0196-9781.

L4 ANSWER 11 OF 100 MEDLINE on STN  
TI Distinct molecular mechanisms for agonist peptide binding to types A and B  
cholecystokinin receptors demonstrated using fluorescence spectroscopy.  
SO Journal of biological chemistry, (2005 Jan 14) 280 (2) 1044-50.  
Electronic Publication: 2004-11-01.  
Journal code: 2985121R. ISSN: 0021-9258.

L4 ANSWER 12 OF 100 MEDLINE on STN  
TI Arrestins and G-protein--coupled receptors: an on-again, off-again  
relationship.  
SO Drug news & perspectives, (1998 Mar) 11 (2) 73-81.  
Journal code: 8809164. ISSN: 0214-0934.

L4 ANSWER 13 OF 100 MEDLINE on STN  
TI Mu-opioid receptor desensitization: is morphine different?.  
SO British journal of pharmacology, (2004 Nov) 143 (6) 685-96. Electronic  
Publication: 2004-10-25. Ref: 70  
Journal code: 7502536. ISSN: 0007-1188.

L4 ANSWER 14 OF 100 MEDLINE on STN  
TI Assays for G-protein-coupled receptor signaling using RGS-insensitive  
Galpha subunits.  
SO Methods in enzymology, (2004) 389 155-69. Ref: 22  
Journal code: 0212271. ISSN: 0076-6879.

L4 ANSWER 15 OF 100 MEDLINE on STN  
TI The pasteurella multocida toxin interacts with signalling pathways to  
perturb cell growth and differentiation.  
SO International journal of medical microbiology : IJMM, (2004 Apr) 293 (7-8)  
505-12. Ref: 49  
Journal code: 100898849. ISSN: 1438-4221.

L4 ANSWER 16 OF 100 MEDLINE on STN  
TI Heterotrimeric G protein subunits are located on rat liver endosomes.  
SO BMC physiology [electronic resource], (2004 Jan 7) 4 1. Electronic  
Publication: 2004-01-07.  
Journal code: 101088687. ISSN: 1472-6793.

L4 ANSWER 17 OF 100 MEDLINE on STN  
TI Conformational states of the corticotropin releasing factor 1 (CRF1)  
receptor: detection, and pharmacological evaluation by peptide ligands.  
SO Peptides, (2003 Dec) 24 (12) 1881-97.  
Journal code: 8008690. ISSN: 0196-9781.

L4 ANSWER 18 OF 100 MEDLINE on STN  
TI Estrogen modulation of G-protein-coupled receptor activation of potassium  
channels in the central nervous system.  
SO Annals of the New York Academy of Sciences, (2003 Dec) 1007 6-16. Ref: 78  
Journal code: 7506858. ISSN: 0077-8923.

L4 ANSWER 19 OF 100 MEDLINE on STN  
TI Heterotrimeric G protein Gi is involved in a signal transduction pathway  
for ATP release from erythrocytes.  
SO American journal of physiology. Heart and circulatory physiology, (2004  
Mar) 286 (3) H940-5. Electronic Publication: 2003-11-13.  
Journal code: 100901228. ISSN: 0363-6135.

L4 ANSWER 20 OF 100 MEDLINE on STN  
 TI Dual inhibition of beta-adrenergic and angiotensin II receptors by a single antagonist: a functional role for receptor-receptor interaction in vivo.  
 SO Circulation, (2003 Sep 30) 108 (13) 1611-8. Electronic Publication: 2003-09-08.  
 Journal code: 0147763. ISSN: 1524-4539.

L4 ANSWER 21 OF 100 MEDLINE on STN  
 TI Identification of two serine residues essential for agonist-induced 5-HT2A receptor desensitization.  
 SO Biochemistry, (2003 Sep 16) 42 (36) 10853-62.  
 Journal code: 0370623. ISSN: 0006-2960.

L4 ANSWER 22 OF 100 MEDLINE on STN  
 TI Mutational uncoupling of alphaA-adrenergic receptors from G proteins also uncouples mitogenic and transcriptional responses in PC12 cells.  
 SO Journal of pharmacology and experimental therapeutics, (2003 Aug) 306 (2) 471-7. Electronic Publication: 2003-04-30.  
 Journal code: 0376362. ISSN: 0022-3565.

L4 ANSWER 23 OF 100 MEDLINE on STN  
 TI C5L2, a nonsignaling C5A binding protein.  
 SO Biochemistry, (2003 Aug 12) 42 (31) 9406-15.  
 Journal code: 0370623. ISSN: 0006-2960.

L4 ANSWER 24 OF 100 MEDLINE on STN  
 TI Endogenous G protein-coupled receptor kinase 6 triggers homologous beta-adrenergic receptor desensitization in primary uterine smooth muscle cells.  
 SO Endocrinology, (2003 Jul) 144 (7) 3058-66.  
 Journal code: 0375040. ISSN: 0013-7227.

L4 ANSWER 25 OF 100 MEDLINE on STN  
 TI G protein-coupled receptor Kinase 2/G alpha q/11 interaction. A novel surface on a regulator of G protein signaling homology domain for binding G alpha subunits.  
 SO Journal of biological chemistry, (2003 Feb 21) 278 (8) 6050-8. Electronic Publication: 2002-11-08.  
 Journal code: 2985121R. ISSN: 0021-9258.

L4 ANSWER 26 OF 100 MEDLINE on STN  
 TI Constitutive activity of G-protein-coupled receptors: cause of disease and common property of wild-type receptors.  
 SO Naunyn-Schmiedeberg's archives of pharmacology, (2002 Nov) 366 (5) 381-416. Electronic Publication: 2002-09-06. Ref: 240  
 Journal code: 0326264. ISSN: 0028-1298.

L4 ANSWER 27 OF 100 MEDLINE on STN  
 TI Apparent loss-of-function mutant GPCRs revealed as constitutively desensitized receptors.  
 SO Biochemistry, (2002 Oct 8) 41 (40) 11981-9.  
 Journal code: 0370623. ISSN: 0006-2960.

L4 ANSWER 28 OF 100 MEDLINE on STN  
 TI Conserved helix 7 tyrosine acts as a multistate conformational switch in the 5HT2C receptor. Identification of a novel "locked-on" phenotype and double revertant mutations.  
 SO Journal of biological chemistry, (2002 Sep 27) 277 (39) 36577-84. Electronic Publication: 2002-07-26.  
 Journal code: 2985121R. ISSN: 0021-9258.

L4 ANSWER 29 OF 100 MEDLINE on STN  
 TI Characterization of cholera toxin B subunit-induced Ca(2+) influx in neuroblastoma cells: evidence for a voltage-independent GM1 ganglioside-associated Ca(2+) channel.  
 SO Journal of neuroscience research, (2002 Sep 1) 69 (5) 669-80.  
 Journal code: 7600111. ISSN: 0360-4012.

L4 ANSWER 30 OF 100 MEDLINE on STN  
 TI Identification of a signal transduction switch in the chemokine receptor CXCR1.  
 SO Journal of biological chemistry, (2002 Aug 30) 277 (35) 31563-6. Electronic Publication: 2002-06-20.  
 Journal code: 2985121R. ISSN: 0021-9258.

L4 ANSWER 31 OF 100 MEDLINE on STN  
 TI Peripheral mechanisms involved with catabolism.  
 SO Current opinion in clinical nutrition and metabolic care, (2002 Jul) 5 (4) 419-26. Ref: 49  
 Journal code: 9804399. ISSN: 1363-1950.

L4 ANSWER 32 OF 100 MEDLINE on STN  
 TI Aripiprazole, a novel antipsychotic, is a high-affinity partial agonist at human dopamine D2 receptors.  
 SO Journal of pharmacology and experimental therapeutics, (2002 Jul) 302 (1) 381-9.  
 Journal code: 0376362. ISSN: 0022-3565.

L4 ANSWER 33 OF 100 MEDLINE on STN  
 TI Effect of pertussis toxin and N-ethylmaleimide on voltage-dependent and -independent calcium current modulation in serotonergic neurons.  
 SO Neuroscience, (2002) 111 (1) 207-14.  
 Journal code: 7605074. ISSN: 0306-4522.

L4 ANSWER 34 OF 100 MEDLINE on STN  
 TI Discrimination of galanin receptor subtypes in RINm5F cells by structurally different galanin radioligands.  
 SO Peptides, (2002 Mar) 23 (3) 545-53.  
 Journal code: 8008690. ISSN: 0196-9781.

L4 ANSWER 35 OF 100 MEDLINE on STN  
 TI Biased inhibition by a suramin analogue of A1-adenosine receptor/G protein coupling in fused receptor/G protein tandems: the A1-adenosine receptor is predominantly coupled to Galpha in human brain.  
 SO Naunyn-Schmiedeberg's archives of pharmacology, (2002 Jan) 365 (1) 8-16.  
 Electronic Publication: 2001-11-07.  
 Journal code: 0326264. ISSN: 0028-1298.

L4 ANSWER 36 OF 100 MEDLINE on STN  
 TI Peptides as receptor ligand drugs and their relationship to G-coupled signal transduction.  
 SO Expert opinion on investigational drugs, (2001 Jun) 10 (6) 1063-73. Ref: 149  
 Journal code: 9434197. ISSN: 1354-3784.

L4 ANSWER 37 OF 100 MEDLINE on STN  
 TI Magnetic fields (MF) of 50 Hz at 1.2 microT as well as 100 microT cause uncoupling of inhibitory pathways of adenylyl cyclase mediated by melatonin 1a receptor in MF-sensitive MCF-7 cells.  
 SO Carcinogenesis, (2001 Jul) 22 (7) 1043-8.  
 Journal code: 8008055. ISSN: 0143-3334.

L4 ANSWER 38 OF 100 MEDLINE on STN  
 TI Molecular scaffold protein and cellular responses.  
 SO Trends in endocrinology and metabolism: TEM, (2001 Jul) 12 (5) 184-6.  
 Journal code: 9001516. ISSN: 1043-2760.

L4 ANSWER 39 OF 100 MEDLINE on STN  
 TI Concomitant increase of G protein-coupled receptor kinase activity and uncoupling of beta-adrenergic receptors in rat myometrium at parturition.  
 SO Endocrinology, (2001 May) 142 (5) 1899-905.  
 Journal code: 0375040. ISSN: 0013-7227.

L4 ANSWER 40 OF 100 MEDLINE on STN  
 TI The cardiac beta-adrenoceptor-G-protein(s)-adenylyl cyclase system in monocrotaline-treated rats.  
 SO Journal of molecular and cellular cardiology, (2000 Dec) 32 (12) 2315-26.  
 Journal code: 0262322. ISSN: 0022-2828.

L4 ANSWER 41 OF 100 MEDLINE on STN  
 TI Selective resistance to parathyroid hormone caused by a novel uncoupling mutation in the carboxyl terminus of G alpha(s). A cause of pseudohypoparathyroidism type Ib.  
 SO Journal of biological chemistry, (2001 Jan 5) 276 (1) 165-71.  
 Journal code: 2985121R. ISSN: 0021-9258.

L4 ANSWER 42 OF 100 MEDLINE on STN  
 TI Studies of the synergistic effect of the Trp/Arg64 polymorphism of the beta3-adrenergic receptor gene and the -3826 A-->G variant of the uncoupling protein-1 gene on features of obesity and insulin resistance in a population-based sample of 379 young Danish subjects.  
 SO Journal of clinical endocrinology and metabolism, (2000 Sep) 85 (9) 3151-4.  
 Journal code: 0375362. ISSN: 0021-972X.

L4 ANSWER 43 OF 100 MEDLINE on STN  
 TI The heat shock cognate protein hsc73 assembles with A(1) adenosine receptors to form functional modules in the cell membrane.  
 SO Molecular and cellular biology, (2000 Jul) 20 (14) 5164-74.  
 Journal code: 8109087. ISSN: 0270-7306.

L4 ANSWER 44 OF 100 MEDLINE on STN  
 TI Thermodynamically distinct high and low affinity states of the A(1) adenosine receptor induced by G protein coupling and guanine nucleotide

ligation states of G proteins.  
SO British journal of pharmacology, (2000 Jun) 130 (3) 595-604.  
Journal code: 7502536. ISSN: 0007-1188.

L4 ANSWER 45 OF 100 MEDLINE on STN  
TI High-affinity binding of urocortin and astressin but not CRF to G  
protein-uncoupled CRFR1.  
SO Peptides, (1999 Nov) 20 (11) 1311-9.  
Journal code: 8008690. ISSN: 0196-9781.

L4 ANSWER 46 OF 100 MEDLINE on STN  
TI Feedback inhibition of G protein-coupled receptor kinase 2 (GRK2) activity  
by extracellular signal-regulated kinases.  
SO Journal of biological chemistry, (1999 Dec 3) 274 (49) 34531-4.  
Journal code: 2985121R. ISSN: 0021-9258.

L4 ANSWER 47 OF 100 MEDLINE on STN  
TI Agonist induced conformation alteration of neurotensin receptor and the  
mechanism behind Na<sup>+</sup> inhibition of 125I-NT binding.  
SO Journal of receptor and signal transduction research, (1999 Nov) 19 (6)  
995-1021.  
Journal code: 9509432. ISSN: 1079-9893.

L4 ANSWER 48 OF 100 MEDLINE on STN  
TI Nurrl, an orphan nuclear receptor, is a transcriptional activator of  
endogenous tyrosine hydroxylase in neural progenitor cells derived from  
the adult brain.  
SO Development (Cambridge, England), (1999 Sep) 126 (18) 4017-26.  
Journal code: 8701744. ISSN: 0950-1991.

L4 ANSWER 49 OF 100 MEDLINE on STN  
TI G-protein coupled receptor kinases as modulators of G-protein signalling.  
SO Journal of physiology, (1999 May 15) 517 ( Pt 1) 5-23. Ref: 186  
Journal code: 0266262. ISSN: 0022-3751.

L4 ANSWER 50 OF 100 MEDLINE on STN  
TI Lesion of septal-hippocampal neurons with 192 IgG-saporin alters function  
of M1 muscarinic receptors.  
SO Neuropharmacology, (1999 Apr) 38 (4) 579-86.  
Journal code: 0236217. ISSN: 0028-3908.

L4 ANSWER 51 OF 100 MEDLINE on STN  
TI Cellular trafficking of G protein-coupled receptor/beta-arrestin endocytic  
complexes.  
SO Journal of biological chemistry, (1999 Apr 16) 274 (16) 10999-1006.  
Journal code: 2985121R. ISSN: 0021-9258.

L4 ANSWER 52 OF 100 MEDLINE on STN  
TI Identification of protein kinase C phosphorylation sites involved in  
phorbol ester-induced desensitization of the histamine H1 receptor.  
SO Molecular pharmacology, (1999 Apr) 55 (4) 735-42.  
Journal code: 0035623. ISSN: 0026-895X.

L4 ANSWER 53 OF 100 MEDLINE on STN  
TI Altered Gq/G11 guanine nucleotide regulatory protein expression in a rat  
model of hepatocellular carcinoma: role in mitogenesis.  
SO Hepatology (Baltimore, Md.), (1999 Feb) 29 (2) 371-8.  
Journal code: 8302946. ISSN: 0270-9139.

L4 ANSWER 54 OF 100 MEDLINE on STN  
TI The cytoplasmic tails of protease-activated receptor-1 and substance P  
receptor specify sorting to lysosomes versus recycling.  
SO Journal of biological chemistry, (1999 Jan 22) 274 (4) 2216-24.  
Journal code: 2985121R. ISSN: 0021-9258.

L4 ANSWER 55 OF 100 MEDLINE on STN  
TI Reciprocal in vivo regulation of myocardial G protein-coupled receptor  
kinase expression by beta-adrenergic receptor stimulation and blockade.  
SO Circulation, (1998 Oct 27) 98 (17) 1783-9.  
Journal code: 0147763. ISSN: 0009-7322.

L4 ANSWER 56 OF 100 MEDLINE on STN  
TI Second site suppressor mutations of a GTPase-deficient G-protein  
alpha-subunit. Selective inhibition of Gbeta gamma-mediated signaling.  
SO Journal of biological chemistry, (1998 Oct 30) 273 (44) 28597-602.  
Journal code: 2985121R. ISSN: 0021-9258.

L4 ANSWER 57 OF 100 MEDLINE on STN  
TI Mutation of a putative amphipathic alpha-helix in the third intracellular  
domain of the platelet-activating factor receptor disrupts receptor/G  
protein coupling and signaling.  
SO Molecular pharmacology, (1998 Mar) 53 (3) 451-8.  
Journal code: 0035623. ISSN: 0026-895X.

L4 ANSWER 58 OF 100 MEDLINE on STN  
 TI Selective uncoupling of RGS action by a single point mutation in the G protein alpha-subunit.  
 SO Journal of biological chemistry, (1998 Mar 6) 273 (10) 5780-4.  
 Journal code: 2985121R. ISSN: 0021-9258.

L4 ANSWER 59 OF 100 MEDLINE on STN  
 TI Gsalpha-selective G protein antagonists.  
 SO Proceedings of the National Academy of Sciences of the United States of America, (1998 Jan 6) 95 (1) 346-51.  
 Journal code: 7505876. ISSN: 0027-8424.

L4 ANSWER 60 OF 100 MEDLINE on STN  
 TI The alpha1B-adrenergic receptor subtype activates the phospholipase C signaling pathway in rat myometrium at parturition.  
 SO Biology of reproduction, (1997 Nov) 57 (5) 1175-82.  
 Journal code: 0207224. ISSN: 0006-3363.

L4 ANSWER 61 OF 100 MEDLINE on STN  
 TI Induction of multiple effects on adenylyl cyclase regulation by chronic activation of the human A3 adenosine receptor.  
 SO Molecular pharmacology, (1997 Oct) 52 (4) 632-40.  
 Journal code: 0035623. ISSN: 0026-895X.

L4 ANSWER 62 OF 100 MEDLINE on STN  
 TI Internalization of the m2 muscarinic acetylcholine receptor. Arrestin-independent and -dependent pathways.  
 SO Journal of biological chemistry, (1997 Sep 19) 272 (38) 23682-9.  
 Journal code: 2985121R. ISSN: 0021-9258.

L4 ANSWER 63 OF 100 MEDLINE on STN  
 TI Somatostatin inhibits interleukin 6 release from rat cortical type I astrocytes via the inhibition of adenylyl cyclase.  
 SO Biochemical and biophysical research communications, (1997 Jun 9) 235 (1) 242-8.  
 Journal code: 0372516. ISSN: 0006-291X.

L4 ANSWER 64 OF 100 MEDLINE on STN  
 TI Order of application determines the interaction between phorbol esters and GTP-gamma-S in dorsal raphe neurons: evidence that the effect of 5-HT is modified upstream of the G protein Ca channel interaction.  
 SO Journal of neurophysiology, (1997 May) 77 (5) 2697-703.  
 Journal code: 0375404. ISSN: 0022-3077.

L4 ANSWER 65 OF 100 MEDLINE on STN  
 TI Oxytocin receptor-mediated activation of phosphoinositidase C and elevation of cytosolic calcium in the gonadotrope-derived alphaT3-1 cell line.  
 SO Endocrinology, (1997 May) 138 (5) 2049-55.  
 Journal code: 0375040. ISSN: 0013-7227.

L4 ANSWER 66 OF 100 MEDLINE on STN  
 TI Mechanisms of beta-adrenergic receptor desensitization: from molecular biology to heart failure.  
 SO Basic research in cardiology, (1996) 91 Suppl 2 29-34. Ref: 62  
 Journal code: 0360342. ISSN: 0300-8428.

L4 ANSWER 67 OF 100 MEDLINE on STN  
 TI Purification and reconstitution of a recombinant human neurokinin-1 receptor.  
 SO Journal of receptor and signal transduction research, (1996 May-Jul) 16 (3-4) 191-207.  
 Journal code: 9509432. ISSN: 1079-9893.

L4 ANSWER 68 OF 100 MEDLINE on STN  
 TI Inhibition of receptor/G protein coupling by suramin analogues.  
 SO Molecular pharmacology, (1996 Aug) 50 (2) 415-23.  
 Journal code: 0035623. ISSN: 0026-895X.

L4 ANSWER 69 OF 100 MEDLINE on STN  
 TI Analysis of granulocyte colony stimulating factor receptor isoforms, polymorphisms and mutations in normal haemopoietic cells and acute myeloid leukaemia blasts.  
 SO British journal of haematology, (1996 Jun) 93 (3) 527-33.  
 Journal code: 0372544. ISSN: 0007-1048.

L4 ANSWER 70 OF 100 MEDLINE on STN  
 TI Lack of discrimination by agonists for D2 and D3 dopamine receptors.  
 SO Neuropsychopharmacology : official publication of the American College of Neuropsychopharmacology, (1995 Jul) 12 (4) 335-45.  
 Journal code: 8904907. ISSN: 0893-133X.

L4 ANSWER 71 OF 100 MEDLINE on STN  
 TI Truncation of the C-terminal tail of the follitropin receptor does not impair the agonist- or phorbol ester-induced receptor phosphorylation and uncoupling.  
 SO Journal of biological chemistry, (1995 Nov 3) 270 (44) 26683-9.  
 Journal code: 2985121R. ISSN: 0021-9258.

L4 ANSWER 72 OF 100 MEDLINE on STN  
 TI Reciprocal binding properties of 5-hydroxytryptamine type 2C receptor agonists and inverse agonists.  
 SO Molecular pharmacology, (1994 Nov) 46 (5) 937-42.  
 Journal code: 0035623. ISSN: 0026-895X.

L4 ANSWER 73 OF 100 MEDLINE on STN  
 TI Antagonists of bradykinin that stabilize a G-protein-uncoupled state of the B2 receptor act as inverse agonists in rat myometrial cells.  
 SO Journal of biological chemistry, (1994 Oct 21) 269 (42) 25970-3.  
 Journal code: 2985121R. ISSN: 0021-9258.

L4 ANSWER 74 OF 100 MEDLINE on STN  
 TI The conserved NPXnY motif present in the gastrin-releasing peptide receptor is not a general sequestration sequence.  
 SO Journal of biological chemistry, (1994 Aug 26) 269 (34) 21755-61.  
 Journal code: 2985121R. ISSN: 0021-9258.

L4 ANSWER 75 OF 100 MEDLINE on STN  
 TI Chimeric G alpha s/G alpha i2 proteins define domains on G alpha s that interact with tubulin for beta-adrenergic activation of adenylyl cyclase.  
 SO Journal of biological chemistry, (1994 Aug 26) 269 (34) 21748-54.  
 Journal code: 2985121R. ISSN: 0021-9258.

L4 ANSWER 76 OF 100 MEDLINE on STN  
 TI Properties of G-protein modulated receptor-adenylyl cyclase system in myocardium of spontaneously hypertensive rats treated with adriamycin.  
 SO International journal of cardiology, (1994 Mar 15) 44 (1) 9-18.  
 Journal code: 8200291. ISSN: 0167-5273.

L4 ANSWER 77 OF 100 MEDLINE on STN  
 TI Identification of a human delta opioid receptor: cloning and expression.  
 SO Life sciences, (1994) 54 (25) PL463-9.  
 Journal code: 0375521. ISSN: 0024-3205.

L4 ANSWER 78 OF 100 MEDLINE on STN  
 TI Second messenger signalling in olfaction.  
 SO Ciba Foundation symposium, (1993) 179 97-109; discussion 109-14, 147-9.  
 Ref: 27  
 Journal code: 0356636. ISSN: 0300-5208.

L4 ANSWER 79 OF 100 MEDLINE on STN  
 TI Continuous spectrofluorometric analysis of formyl peptide receptor ternary complex interactions.  
 SO Molecular pharmacology, (1994 Jan) 45 (1) 65-73.  
 Journal code: 0035623. ISSN: 0026-895X.

L4 ANSWER 80 OF 100 MEDLINE on STN  
 TI Guanine nucleotide binding proteins mediate D2 dopamine receptor activation of a potassium channel in rat lactotrophs.  
 SO Journal of physiology, (1993 Mar) 462 563-78.  
 Journal code: 0266262. ISSN: 0022-3751.

L4 ANSWER 81 OF 100 MEDLINE on STN  
 TI Photoaffinity labelling and radiation inactivation of the leukotriene B4 receptor in human myeloid cells.  
 SO European journal of pharmacology, (1993 Jan 15) 244 (2) 161-73.  
 Journal code: 1254354. ISSN: 0014-2999.

L4 ANSWER 82 OF 100 MEDLINE on STN  
 TI Regulation of spontaneous opening of muscarinic K+ channels in rabbit atrium.  
 SO Journal of physiology, (1991 Feb) 433 589-613.  
 Journal code: 0266262. ISSN: 0022-3751.

L4 ANSWER 83 OF 100 MEDLINE on STN  
 TI Catabolism of hemoglobin-haptoglobin complex in microsome subfractions.  
 SO Chemical & pharmaceutical bulletin, (1992 Jul) 40 (7) 1847-51.  
 Journal code: 0377775. ISSN: 0009-2363.

L4 ANSWER 84 OF 100 MEDLINE on STN  
 TI Occupancy of G alpha s-linked receptors uncouples chemoattractant receptors from their stimulus-transduction mechanisms in the neutrophil.  
 SO Blood, (1992 Aug 15) 80 (4) 1052-7.  
 Journal code: 7603509. ISSN: 0006-4971.

L4 ANSWER 85 OF 100 MEDLINE on STN  
 TI Ligand-mediated internalization of glucagon receptors in intact rat liver.  
 SO Endocrinology, (1992 Jul) 131 (1) 447-57.  
 Journal code: 0375040. ISSN: 0013-7227.

L4 ANSWER 86 OF 100 MEDLINE on STN  
 TI GAP domains responsible for ras p21-dependent inhibition of muscarinic  
 atrial K<sup>+</sup> channel currents.  
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FILE 'MEDLINE' ENTERED AT 13:49:44 ON 10 JAN 2006

L1 15992 S UNCOUPL?  
 L2 2638 S L1(P) RECEPTOR#  
 L3 780 S L2(P)G  
 L4 100 S L3(P) IDENT?

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L5 1 PERTIS?

=> s pertus?

L6 20722 PERTUS?

=> s 16(p)l1

L7 216 L6(P)L1

=> s 16(p)l2

L8 193 L6(P)L2

=> s 16(p)l3

L9 143 L6(P)L3

=> d 1-143 ti so

L9 ANSWER 1 OF 143 MEDLINE on STN  
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L3: Entry 92 of 99

File: USPT

Mar 4, 2003

US-PAT-NO: 6528271

DOCUMENT-IDENTIFIER: US 6528271 B1

TITLE: Inhibition of .beta.arrestin mediated effects prolongs and potentiates  
opioid receptor-mediated analgesia

DATE-ISSUED: March 4, 2003

## INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
Bohn; Laura M.	Durham	NC		
Lin; Fang-Tsyr	Durham	NC		
Caron; Marc G.	Hillsborough	NC		
Lefkowitz; Robert J.	Durham	NC		

## ASSIGNEE-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY	TYPE CODE
Duke University	Durham	NC			02

APPL-NO: 09/469554 [\[PALM\]](#)

DATE FILED: December 22, 1999

## PARENT-CASE:

This application is a continuation-in-part of U.S. application Ser. No. 09/233,530, now issued as U.S. Pat. No. 6,110,693, filed Jan. 20, 1999; which is a continuation of U.S. application Ser. No. 08/869,568, now issued as U.S. Pat. No. 5,891,646, filed Jun. 5, 1997.

INT-CL-ISSUED: [07] [C07 K 14/00](#), [C07 K 17/00](#), [C12 Q 1/00](#), [G01 N 33/53](#), [G01 N 33/567](#)

US-CL-ISSUED: 435/7.2; 435/4, 530/350

US-CL-CURRENT: [435/7.2](#); [435/4](#), [530/350](#)

FIELD-OF-CLASSIFICATION-SEARCH: 424/9.1, 424/9.2, 435/4, 435/7.2, 530/350  
See application file for complete search history.

## PRIOR-ART-DISCLOSED:

## U.S. PATENT DOCUMENTS

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<input type="checkbox"/>	<u>5821067</u>	October 1998	Grandy et al.	435/7.2
<input type="checkbox"/>	<u>5882944</u>	March 1999	Sadee	436/501
<input type="checkbox"/>	<u>5891646</u>	April 1999	Barak et al.	435/7.2
<input type="checkbox"/>	<u>6007986</u>	December 1999	Sadee	435/6
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<input type="checkbox"/>	<u>6096756</u>	August 2000	Crain et al.	514/282
<input type="checkbox"/>	<u>6103492</u>	August 2000	Yu	435/69.1
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FOREIGN-PAT-NO	PUBN-DATE	COUNTRY	CLASS
WO98/55635	December 1998	WO	

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ART-UNIT: 1632

PRIMARY-EXAMINER: Baker; Anne-Marie

ATTY-AGENT-FIRM: Burns, Doane, Swecker & Mathis, L.L.P.

ABSTRACT:

The present invention provides a  $\beta$ -arrestin knockout mouse useful for screening compounds for efficacy in controlling pain, methods of controlling pain in subjects by inhibiting binding of  $\beta$ -arrestin to phosphorylated  $\mu$  opioid receptors, and methods of screening a compound for activity in potentiating  $\mu$  opioid receptor agonist activity (e.g., morphine activity) by determining whether or not said compound inhibits  $\beta$ -arrestin binding to a phosphorylated  $\mu$  opioid receptor.

12 Claims, 8 Drawing figures

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L6: Entry 1 of 3

File: PGPB

Apr 14, 2005

DOCUMENT-IDENTIFIER: US 20050079178 A1

TITLE: Inhibitor of cardiac tachyarrhythmias

Detail Description Paragraph:

[0105] Desensitization of G-protein receptors, including .beta.-adrenergic, adenosine, muscarinic, and angiotensin receptors, occurs by: 1) ligand-independent phosphorylation through protein kinase A and 2) ligand-dependent phosphorylation through .beta.-adrenergic receptor kinase. It is proposed that increased .beta.-adrenergic receptor sensitivity and increased isoproterenol-stimulated c-AMP formation results directly from decreased .beta.-adrenergic receptor kinase-1 expression/activity (.beta.-ARK-1) within injured epicardium. .beta.-ARK-1, by phosphorylating ligand-activated .beta.-adrenergic receptors, desensitizes the .beta.-adrenergic receptor and precipitates its internalization from the cell membrane to the cell interior by an obligate transport protein, .beta.-arrestin (Pitcher et al., 1998; Bunemann et al., 1999; Krupnick et al., 1998). .beta.ARK-1, a G-receptor kinase of the superfamily of G-receptor inactivating enzymes, is the principal mechanism providing for the desensitization and the termination of .beta.-adrenergic receptor stimulation in myocardium and other tissues (Pitcher et al., 1998; Bunemann et al., 1999; Krupnick et al., 1998). The decrease of .beta.-ARK-1 in injured myocardium has been determined by direct measurement of myocardial .beta.-ARK-1 activity (FIG. 12), immunoblot of .beta.-ARK-1 expression (FIG. 13A), and immunohistofluorescence of myocardial .beta.-ARK-1 content. Only a partial recovery of .beta.-ARK-1 activity was observed at 96 hours (FIG. 13B). It appears that the decrease in .beta.-ARK-1 in ischemically-injured myocardium results from an increased degradation of .beta.-ARK-1 in ischemically-injured epicardium.

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L3: Entry 34 of 99

File: PGPB

May 27, 2004

DOCUMENT-IDENTIFIER: US 20040101887 A1

TITLE: Methods of assaying receptor activity and constructs useful in such methods

Summary of Invention Paragraph:

[0004] It has been postulated that members of the GPCR superfamily desensitize via a common mechanism involving G protein-coupled receptor kinase (GRK) phosphorylation followed by arrestin binding! Gurevich et al., J. Biol. Chem. 270:720 (1995); Ferguson et al., Can. J. Physiol. Pharmacol. 74:1095 (1996). However, the localization and the source of the pool of arrestin molecules targeted to receptors in response to agonist activation was unknown. Moreover, except for a limited number of receptors, a common role for .beta.-arrestin in GPCR desensitization had not been established. The role of .beta.-arrestins in GPCR signal transduction was postulated primarily due to the biochemical observations.

Detail Description Paragraph:

[0036] Phosphorylation of GPCRs is a mechanism leading to desensitization of the receptors; receptors that have been continuously or repeatedly stimulated lose responsiveness, whereas the responses of other receptors remain intact. See Harden, Pharmacol. Rev. 35:5 (1983); Benovic et al., Annu. Rev. Cell. Biol. 4:405(1988). In a variety of cells, specific kinases have evolved for specific GPCRs. Desensitization occurs via the following pathway: agonist occupancy of the receptor transforms the receptor into all appropriate substrate for an associated kinase; .beta.-arrestin binds to the kinase phosphorylated receptor and prevents subsequent interaction with the appropriate G-protein, as well as initiating both internalization and resensitization processes. Ferguson et al, Science, 271:363 (1996); Lohse et al., Science 248:1547 (1990). .beta.-arrestin dependent desensitization is induced only when the GPCR is activated by ligand binding, and is an example of homologous desensitization (i.e., the ligand desensitizes only its target receptors). Lohse et al. (1990) and Attramadal et al., J. Biol. Chem. 267:17882 (1992) provide cDNA and amino acid sequences of .beta.-arrestin. Various isoforms of .beta.-arrestin are known; as used herein, .beta.-arrestin refers to all such isoforms of .beta.-arrestin, proteins having substantial sequence similarity thereto which are functional .beta.-arrestins, and functional fragments thereof. Functional fragments of .beta.-arrestin, its isoforms and analogs, may be determined using techniques as known in the art.

Detail Description Paragraph:

[0066] The present methods may further be used to assess or study the effects of any molecule in the GPCR pathway which exerts its effect upstream of .beta.-arrestin binding (i.e., prior to .beta.-arrestin binding to the phosphorylated GPCR). Thus the present invention provides methods for assessing GPCR pathway functions in general. As used herein, the GPCR pathway refers to the series of events which starts with agonist activation of a GPCR followed by desensitization of the receptor via G protein-coupled receptor kinase (GRK) phosphorylation and .beta.-arrestin binding.

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L3: Entry 52 of 99

File: PGPB

Aug 21, 2003

DOCUMENT-IDENTIFIER: US 20030157553 A1

TITLE: Methods of assaying for G protein-coupled receptor ligands and modulators

Summary of Invention Paragraph:

[0004] Signaling by diverse GPCR agonists is believed to be terminated by a uniform two-step mechanism (Freedman and Lefkowitz (1996) Recent Prog. Horm. Res. 51:319-353). According to the model, activated receptor is first phosphorylated by a G protein-coupled receptor kinase (GRK). An arrestin protein then binds to the activated phosphoreceptor, thereby blocking G protein interaction. Arrestin-receptor complex is subsequently internalized, whereupon receptor is either dephosphorylated and recycled back to the plasma membrane (resensitization) or sorted to lysosomes and destroyed (down-regulation). Thus, the formation of the arrestin-receptor complex appears to be the final step of desensitization and the first step of resensitization and/or receptor down-regulation.

Summary of Invention Paragraph:

[0005] GPCRs are activated by an extremely wide variety of external stimuli and are now believed to play a role in regulating the activity of virtually all eukaryotic cells. In contrast, the repertoire of receptor kinases and arrestins involved in the desensitization of these receptors is rather limited: six GRKs and four arrestins have thus far been found in mammals (reviewed in Freedman and Lefkowitz, supra).

Summary of Invention Paragraph:

[0006] The fact that a limited number of receptor kinases and arrestins can regulate desensitization of numerous GPCRs makes these molecules attractive targets for research designed to identify potential modulators of GPCR signaling, in particular modulators of such receptors for use as human therapeutics. The present invention is based, at least in part, on a new use for phosphorylation-independent mutants of arrestin proteins as assay components in screening assays designed to identify ligands and/or modulators of G protein-coupled receptors (GPCRs). In particular, it has been discovered that phosphorylation-independent mutants of arrestin capable of binding GPCRs in a manner independent of the natural requirement for phosphorylation by intracellular receptor kinases. Such phosphorylation-independent mutants of arrestin retain there dependence on agonist for GPCR-binding, however. As such, phosphorylation-independen- t arrestin mutants are particularly well-suited for in vitro assays, for example, in assays that are directed to the identification of natural and surrogate agonists of orphan GPCRs (e.g., of known and/or orphan GPCRs, for example, in orphan GPCR ligand fishing assays). The phosphorylation-independent mutants of arrestin are also particularly well-suited for in vitro assays that are directed to the identification of GPCR antagonists and/or agonists.

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L3: Entry 52 of 99

File: PGPB

Aug 21, 2003

PGPUB-DOCUMENT-NUMBER: 20030157553

PGPUB-FILING-TYPE: new

DOCUMENT-IDENTIFIER: US 20030157553 A1

TITLE: Methods of assaying for G protein-coupled receptor ligands and modulators

PUBLICATION-DATE: August 21, 2003

## INVENTOR-INFORMATION:

NAME

CITY

STATE

COUNTRY

Berstein, Gabriel

Woburn

MA

US

APPL-NO: 09/800137 [\[PALM\]](#)

DATE FILED: March 5, 2001

## RELATED-US-APPL-DATA:

Application is a non-provisional-of-provisional application 60/186706, filed March 3, 2000,

INT-CL-PUBLISHED: [07] [G01](#) [N](#) [33/53](#)

US-CL-PUBLISHED: 435/7.1

US-CL-CURRENT: [435/7.1](#)

REPRESENTATIVE-FIGURES: NONE

## ABSTRACT:

The present invention features methods of assaying for ligands and/or modulators of G protein-coupled receptors, for example, orphan G protein-coupled receptors. The methods of the invention feature constitutively active arrestin mutants, in particular, phosphorylation independent mutants. Also described are specific phosphorylation independent arrestin mutants and methods of making such mutants.

## RELATED APPLICATIONS

[0001] This application claims the benefit of prior-filed provisional patent application U.S. Ser. No. 60/186,706, entitled "Methods of Assaying for G Protein-Coupled Receptor Ligands and Modulators", filed Mar. 3, 2000 (pending). The content of the above referenced application is incorporated herein in its entirety by this reference.

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L7: Entry 1 of 1

File: PGPB

May 23, 2002

DOCUMENT-IDENTIFIER: US 20020062490 A1

TITLE: Hypertension

Summary of Invention Paragraph:

[0004] A functional abnormality leading to hypertension can reflect an imbalance between G protein-coupled receptor (GPCR) mediated vasoconstriction and vasodilation. GPCR signaling is mediated via heterotrimeric G proteins which, following receptor activation, dissociate and release activated G.alpha. and G.beta..gamma. subunits, both of which are capable of molecular signaling (Post et al, FASEB J. 10:741-749 (1996)). One G protein family member, Gq, is activated by several GPCRs mediating vasoconstriction and its activation can dramatically increase systemic vascular resistance (SVR), mean arterial pressure (MAP) and myocardial and vascular hypertrophy. Activation of another member of the G protein family, Gs, by GPCRs including .beta.-adrenergic receptors (ARs), can mediate vasodilation and a decrease in SVR and MAP. Impairment in .beta.AR-mediated vasodilation due to an alteration in receptor/G-protein coupling increases SVR and has been described to occur in both human and animal models of hypertension (Feldman, J. Clin. Invest. 85:647-652 (1990); Feldman et al, Hypertension 26:725-732 (1995)). This defective .beta.AR coupling is accompanied by selective increases in expression and activity of the G protein-coupled receptor kinase 2 (GRK2 or .beta.ARK1) which has been found in the lymphocytes of hypertensive patients (Gros et al, J. Clin. Invest. 99:2087-2093 (1997)). GRK2 phosphorylates and desensitizes agonist-occupied GPCRs, including BARs, and therefore prevents activation of G proteins. Of the 6 members of the GRK family, GRK2, GRK3, GRK5 and GRK6 have been found in VSM (Gros et al, J. Clin. Invest. 99:2087-2093 (1997); Ishizaka et al, J. Biol. Chem. 272:32482-32488 (1997)). Although none of these kinases show substrate selectivity in vitro, recent evidence suggests that in vivo in the heart they show specific actions (Eckhart et al, Circ. Res. 86:43-50 (2000); Koch et al, Ann. Rev. Physiol. 62:237-260)). With respect to important vascular GPCRs, GRK2 desensitizes the .beta..sub.2AR and angiotensin II receptor in vivo, whereas it is ineffective on the .alpha..sub.1BAR (Eckhart et al, Circ. Res. 86:43-50 (2000); Koch et al, Ann. Rev. Physiol. 62:237-260)).

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L3: Entry 18 of 99

File: PGPB

Feb 10, 2005

DOCUMENT-IDENTIFIER: US 20050032158 A1

TITLE: Binding assays that use human T1R2 to identify potential taste modulators

Detail Description Paragraph:

[0077] "Inhibitors," "activators," and "modulators" of T1R genes or proteins are used interchangeably to refer to inhibitory, activating, or modulating molecules identified using in vitro and in vivo assays for taste transduction, e.g., ligands, agonists, antagonists, and their homologs and mimetics. Inhibitors are compounds that, e.g., bind to, partially or totally block stimulation, decrease, prevent, delay activation, inactivate, desensitize, or down regulate taste transduction, e.g., antagonists. Activators are compounds that, e.g., bind to, stimulate, increase, open, activate, facilitate, enhance activation, sensitize, or up regulate taste transduction, e.g., agonists. Modulators include compounds that, e.g., alter the interaction of a receptor with: extracellular proteins that bind activators or inhibitor (e.g., ebnerin and other members of the hydrophobic carrier family); G proteins; kinases (e.g., homologs of rhodopsin kinase and beta adrenergic receptor kinases that are involved in deactivation and desensitization of a receptor); and arrestins, which also deactivate and desensitize receptors. Modulators can include genetically modified versions of T1R family members, e.g., with altered activity, as well as naturally occurring and synthetic ligands, antagonists, agonists, small chemical molecules and the like. Such assays for inhibitors and activators include, e.g., expressing T1R family members in cells or cell membranes, applying putative modulator compounds, in the presence or absence of taste stimuli, e.g., sweet taste stimuli, and then determining the functional effects on taste transduction, as described above. Samples or assays comprising T1R family members that are treated with a potential activator, inhibitor, or modulator are compared to control samples without the inhibitor, activator, or modulator to examine the extent of modulation. Control samples (untreated with modulators) are assigned a relative T1R activity value of 100%. Inhibition of a T1R is achieved when the T1R activity value relative to the control is about 80%, optionally 50% or 25-0%. Activation of a T1R is achieved when the T1R activity value relative to the control is 110%, optionally 150%, optionally 200-500%, or 1000-3000% higher.

Detail Description Paragraph:

[0201] Activated GPCR receptors become substrates for kinases that phosphorylate the C-terminal tail of the receptor (and possibly other sites as well). Thus, activators will promote the transfer of 32P from gamma-labeled GTP to the receptor, which can be assayed with a scintillation counter. The phosphorylation of the C-terminal tail will promote the binding of arrestin-like proteins and will interfere with the binding of G proteins. The kinase/arrestin pathway plays a key role in the desensitization of many GPCR receptors. For example, compounds that modulate the duration a taste receptor stays active would be useful as a means of prolonging a desired taste or cutting off an unpleasant one. For a general review of GPCR signal transduction and methods of assaying signal transduction, see, e.g., Methods in Enzymology, vols. 237 and 238 (1994) and volume 96 (1983); Bourne et al., Nature, 10:349:117-27 (1991); Bourne et al., Nature, 348:125-32 (1990); Pitcher et al., Annu. Rev. Biochem., 67:653-92 (1998).

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